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Clinical Proceedings

OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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(1) Diamond, L. K.; Smith, N. J., and Vaughan, V. C., III, in Nelson, W. E.: Textbook of Pediatrics, ed. 6, Philadelphia, W. B. Saunders Company, 1954, p. 962. (2) Niccum, W. L.; Jackson, R. L., and Stearns, G.: A.M.A. Am. J. Dis. Child. 86:553, 1953.

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CLINICAL PROCEEDINGS
OF THE CHILDREN'S HOSPITAL

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COMMON FOOT PROBLEMS IN INFANTS AND CHILDREN

A Panel Discussion

Frederic G. Burke, M.D.,* Allen S. Lloyd, M.D.,† William S. Anderson, M.D.,‡ William J. Tobin, M.D.§

Dr. Burke:

It has been said that "position in life is everything". It has been well established that the position of the infant's legs in the womb, in addition to their disposition in the early weeks and months of post-natal life plays a considerable role in the enhancement of so-called foot problems of early life. Many healthy, perfectly normal children sleep on their abdomen with their rump in the air and their feet tucked under. This, for example has been demonstrated to be a mechanical factor which tends to enhance the adductor varus disposition of the foot. However, I would say that to disturb the normal sleeping pattern of such a baby up to the age of 6 to 8 months, with the purpose in mind of avoiding such influence when in every other sense the infant is doing well, would be malpractice. Whether such sleeping habits can easily be changed at a later date is a different matter and I should like to hear a comment or two from the panel on this point. In the same vein the "television squat", in which position the feet are everted in an oriental style is noted as a new hazard in the development of the feet of the child and one which would appear to lend itself to easy correction. Encouragement of these children to accept chairs when viewing television would promise to keep a few more patients from needing the manipulations of an orthopedic specialist.

I would also like to comment on the medical environmental influence as a factor influencing the incidence of "foot problems". In some hospitals one or two staff members may be extremely enthusiastic, indeed over enthusiastic, in tackling so-called foot problems in infants. The other day I was talking to an orthopedist as he was applying an evertor cast for a true adductor varus on a 3 month old baby when a student nurse from an outlying hospital commented, "This is the first one of these that I have seen since I have been here, but in my own hospital I saw fifteen of these casts applied in six months in the newborn nursery". In her hospital it would appear that 10 per cent of newborn babies were having casts put on their feet

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before they left the hospital. It would appear that at least in that particular hospital there was not a full awareness of the salutary effects of time. The natural intrauterine position of the majority of infants is such that the feet are usually in adductor varus position. Of course, the opposite can occasionally occur, and this is a more pathological situation, particularly in twins or in babies of breech deliveries where there might be a valgus deformity.

In regard to knock-knees, I would say that many of these children with mild degrees of genu valgus and even some with more marked degrees, particularly overweight children, have been encouraged to walk too early, that is, they are given arch supported, hard soled shoes the first time they stand up at 8 or 9 months. The firmer support given by the hard sole is an encouragement, in my opinion, to have infants walk earlier than they are physiologically or anatomically prepared to. This is particularly true between the eighth and the fourteenth month, when many babies have been overfed and are in the overweight group. Until the physiological slimming process, due to continued height growth with diminished acceleration of weight gain occurs, usually between the ages of 14 and 24 months, I do not believe that these children should be encouraged to walk any earlier than they want to by themselves. I would like to hear this point commented upon by the panel.

Finally, a comment or two upon what constitutes normal walking: It is well recognized in the toddler, between the ages of 14 and 18 months that eversion of the feet is normal and usually is more evident in the right than the left foot in right-handed children. Thus this situation of having one foot appear to drag and giving an apparent limp, is one of the normal phenomena for which counsel and reassurance must be given to parents. Heel-toe walking usually does not come about until 18 months.

Just as we examine eye, ear, nose, and throat, so attention to the position of the feet, particularly when the infant starts to stand, is of considerable importance. And, of course, it is just as important to learn to differentiate normal variations from those situations which call for prompt and early management with orthopedic consultation. With these problems in mind, I would like to call upon Dr. Lloyd to give us some of his opinions.

Dr. Lloyd:

On the question of treatment for metatarsus adductus (swinging in of the big toe is the most common thing), it is a matter of judgment as to which feet have a metatarsus primus adductus and which ones do not. Of those which have it I have never seen one clear up spontaneously. I have seen children with a foot that tended to turn in, but which can be easily pushed into over-correction. That is not a metatarsus adductus. Incidentally, many of those cases do not occur at birth or in the early weeks of life,

and the parent may come in with a child a year and a half or two years old, somewhat put out because the pediatrician had not discovered the condition until the last visit. Why in the world did he not notice this when that child was born? The answer is that in many of those cases it does not appear when the child is born. I have seen it in children for whom I have treated one foot for another condition and looked at both feet when they came in, with records there to show that they were normal. Six months to a year and a half later, an occasional one of these children has a metatarsus adductus he never showed previously. So frankly, if they really have a metatarsus adductus, I think very definitely they should be treated, with shoes in mild cases or with plaster in the more severe cases.

As for the pronated feet, I will just tell you what I tell my patients, and that is all I know about it. When I look at a pronated foot in a child, nine times out of ten I do not know whether God is going to correct that thing or not. If He is going to, you can not conceivably do the child any harm by having a support under the arch and a wedge on the inner side of the heel, to prevent more pronation from going on. I tell them that with all the corrections in the world some of those feet at the end of 12 years will be just as pronated as they were to begin with. Personally, I do not think shoes ever cure pronation. The reason I put on these shoes is that in the cases where God is going to give them an arch anyway to correct the pronation, I can not do them any harm by putting on shoe corrections. In the second place, if He is not going to give them an arch, I have at least held the bones and ligaments in a position where they are not completely and always in an abnormal position. Then when these youngsters are 14 or 15 years of age and I give them a method of walking which automatically holds up their arch, they can do it with a great deal more ease than they could if I had let them go all those years with the foot constantly in an abnormal position.

Dr. Burke:

What age are you referring to in that group?

Dr. Lloyd:

Anywhere from a year on, whenever the parents first bring them in. I might be able to let a child go along without any correction and he might get perfectly all right. On the other hand, since he may not correct spontaneously, I would rather treat him in a harmless manner so that if he does not develop the arch, at least the foot will be more comfortable in the normal position, and it will be easier for him to walk in the manner that I teach him later on.

As far as the bowlegs and knock-knees are concerned, the one thing that

sticks with me is information in publications by Nacklas in Baltimore. He pointed out that it is normal for a child to be born with internal tibial torsion and with bowlegs, and normally by the time the child gets to the age of from 2 to 2½ years, the fat and soft tissue over the medial femoral condyles will be more prominent than it has been. Usually from that age until the the age of 5 to 5½ years the children are getting more knock-kneed. Then they start to straighten out and they end up where ever that individual is going to end up. Therefore, if I see a bowlegged child within what Nacklas calls the bowlegged age group, up to 2½ years, I simply observe him. If there is not a gross deformity that obviously needs treatment, I simply give some psychological treatment with a little correction on the shoe that can not possibly do any harm and measure the distance between the knees. In many of those cases, as they are measured every three months or so, they gradually straighten out, and if they are becoming less bowlegged you know you do not have to do anything about them. The same thing applies to knock-knees.

We had a little guy in the clinic with bowlegs sufficiently bad that we took photographs of him in addition to his x-rays. We advised the mother that he should have braces, but she did not bring him back. We saw him approximately 2½ years later when she brought him back for us to treat his knock-knees. He had had no treatment whatever in the meantime. He had gone from an extreme bowleg to an extreme knock-knee. I do not mean that happens in every case, but it can happen and it illustrates the change from a bowleg to a knock-knee without any treatment at all. Therefore, it is only those degrees of bowleg or knock-knee that are abnormal within their age group that I feel like treating actively. You always have to treat the parents, of course.

Dr. Burke:

I would like to hear Dr. Anderson's comments and questions that he might have both on your discourse and from his own observations.

Dr. Anderson:

When I was a resident my chief tried to persuade me unsuccessfully to go down and work in the orthopedic clinic during the final year of my residency. In the folly of my youth I thought there were too many other interesting things on the ward, and I regret many times that I did not do so because when you do get in practice, as Dr. Burke has said, these minor foot variations are quite a headache. I would agree completely on the uterine positional comments that Dr. Burke has made. Dr. Charles C. Chapple in Philadelphia has shown over and over again that soon after birth, if a baby is unhappy and crying, if you will get him back into the uterine position, he will go right off to sleep again. Most of those babies at

first look as if they are bowlegged and they also look as if they have a metatarsus varus. Most of that is uterine positional, and in the course of several months if it does not correct itself, then the question arises as to whether the orthopedist should see it. Fortunately, as we all know, old Mother Nature is a very good doctor, particularly as far as feet go, and if you leave the feet alone a great many of them will get perfectly all right.

The same thing applies to Dr. Lloyd's comments on flatfoot or pronation. I do not quite agree with him on the one year. As you all know, there is a fat pad in the arch of the foot for at least the first year, and I personally do not think that at a year you can tell what the arch of that child is going to be at 2 years of age. I prefer personally to wait until the child is 2 years of age before attempting to evaluate the arch. I would like to hear Dr. Tobin's comment on that too.

I would also like to hear Dr. Tobin comment on the question of the high versus the low shoes. I think that is quite an individual opinion. Pediatricians would probably be divided half and half as to whether they would start off a child in a low shoe or a high shoe. I disagree a little bit with Dr. Burke. I do not believe putting a baby in a hard shoe makes him walk any sooner than he would in a soft shoe. I prefer personally a high shoe with a very thin, flexible sole, and a moccasin seam in the toe to keep the box toe after the baby wets the shoe a great many times. It keeps it a little bit from contracting down on the toe.

The other thing that I would like Dr. Tobin or Dr. Lloyd to comment about is whether foot exercises are of any value whatsoever. An arch support and a Thomas heel remind me of putting an arm in a sling; it gives support to the arch but it does not improve the tone of the arch, and I wonder whether exercise would have a place here.

There is a great hereditary factor in pronation which I think should be mentioned just for emphasis. The knock-knees and the bowlegs I want to mention because I think the physiological bowing worries mothers a great deal. I think that one of the most common questions we are asked before the baby starts to stand is: "Is my baby bowlegged?"

The other thing that has not been mentioned is the question of overlapping toes. That is another question that mothers bring to our attention and worry about. I would like the orthopedists to comment on that.

As far as shoes go, I would like to hear, just for interest, the orthopedist's comment on cowboy boots and "Jumping-Jacks", both of which I think are equally bad.

Dr. Burke:

I think from experience in raising my own youngsters that one very practical reason that a high-top shoe should be used until about the second year is the rapidity with which your shoe bills go up when low tops are

used. The high-top shoes have one big virtue, the children can not take them off. Incidentally, I do not feel that the high part of the high top shoe gives any real ankle support, because the support is supplied by the understructure of the shoe.

Dr. Anderson:

We agree completely, and I think your point, one I failed to make, about the economic factor is one of the most important.

Dr. Burke:

As far as the hereditary feature, I have heard an interesting explanation that those groups, particularly the Semites, who have lived in hilly country and whose ancestors have been climbing hills for thousands and thousands of years, do not climb such hills in a heel-toe position but normally in a pronated position. From a developmental viewpoint this may be very practical. It should be noted that many such pronated feet are perfectly adequate and functional. I am sure many of these people marched just as far in the army and carry on their normal functions without obvious complaint. Dr. Tobin, would you like to take up this discussion?

Dr. Tobin:

I hardly know where to begin, but I do know where to stop. You know, when we have been talking about the feet and knees I presume that in the minds of all of you our discussion more or less stops with the feet and knees. But we can not stop there. We have got to consider the child's over-all posture. On the basis of any foot or knee deformities, there are bound to be changes in femoral torsion; the knee may point in; there may be changes in the hips and in the spine. I realize that is not the subject under discussion here, but I did want to mention it.

Regarding the forefoot adduction, Dr. Lloyd brought out the primus metarsus varus, which is the classical name of the pigeon-toe type of foot in which the great toe stands out separately in an adduction position from the rest of the toes which are fairly straight. For the sake of definition that is not the same as forefoot adduction which is actually one-third of a club foot. These cases are almost invariably associated with tibial torsion. It has been brought out, and I wish to emphasize the fact that these are retentions of the intra-uterine position, particularly in the third trimester of development when the baby is like the chick in the egg, in "cramped quarters". There is a marked variability in all the deformities and abnormalities that everyone has mentioned. I think it is up to the pediatrician, on the basis of his knowledge and experience, to decide which is not within the normal scope of variation. I do not agree entirely with Dr. Burke,

unless I misunderstood him, that the cause of a lot of this turning in may be the result of the child being up on his knees and his rump with his feet turned in. I think if a child has an abnormal amount of tibial torsion, or forefoot adduction, he is going to turn his feet in, and although that position may be physiological for the babies that sleep on their tummies, it certainly is going to aggravate the condition. This again is a matter of degree. If it is not bad, I agree with Dr. Anderson and the others that Mother Nature is very kind to all of us, particularly to children, and a lot of these conditions will improve.

Another interesting variation is turning in or out of the foot due to hip problems. In the intra-uterine stage the legs may go one of three ways: sometimes you see kiddies born with an extreme degree of external rotation of both lower extremities, which is unusual and is due to tightness of the external rotator muscles of the hip, or one leg may be turned in and the other one turned out, or thirdly, both are turned in.

There is one other bowleg problem that has not been brought up that I think we should mention. Dr. Burke said that he did not want us to take up the osseous abnormalities, but I do want to mention tibia vara, or osteochondrosis of the upper tibial epiphysis, which gives a bowing of the lower leg due to an angulation just below the knee.

Dr. Burke:

Do you mean Blount's disease?

Dr. Tobin:

Yes.

Dr. Burke:

We have not seen a case of Blount's disease in ten years, even carefully looking for it radiologically. Is it common in your experience?

Dr. Tobin:

No, not so common as the physiological bowleg, I agree with you, but I am trying to bring out the point that there is a difference tetween the bowing that is physiological and the angulation that one sees in Blount's disease. I really think Blount's disease is more common than you infer. I certainly see it.

Dr. Burke:

I am sure it exists, but I think that of all the osteochrondritides it is probably one of the most uncommon.

Dr. Tobin:

Another point is that these cases in the past have been considered being rachitic in origin, but it is rather embarassing even to imply the existence of rickets in the present day care of infants.

Let us consider some of the specific questions. The overlapping toe is oftentime a strictly hereditary feature and if you will go back and ask the mother and the grandmother you may find that they too have had an overlapping fifth toe. There is not much one can do for them. We also see at times a rotation of the toes in which strapping of the toes together with a derotation is rarely helpful.

I do not wholly agree with Dr. Lloyd in his attitude about the flat feet, because there is one thing we must remember. Pronation, or flatness of the foot, and knock-knees are mechanically associated just as tibial torsion, forefoot adduction and bowlegs. Although we do not get excited about some of the relaxation of the arches in infants. In those whose internal malleoli are four to six inches apart, should be treated, because the increasing amount of pronation will only be followed by increasing knock-knee deformity.

Regarding high versus low shoes, it is too bad perhaps in a way that we have to wear shoes at all. The southern Negro has a big, broad, flat foot that serves him well. The African natives do not go around wearing shoes. People with that type of flat feet have no trouble at all. Perhaps we are in one sense of the word victims of circumstances and civilization as we put shoes on feet that could function perfectly well without them.

Dr. Burke:

They certainly tend to shorten or prevent full development of the gastrocnemius tendon.

Dr. Tobin:

Well, yes, but a tight heel cord is another thing to be brought up, because occasionally we see a child who, for no apparent reason except perhaps some obscure neurological condition, comes in with bilateral tightness of the heel cords. Certainly a child like that you would not want to put in high-heeled shoes.

Incidentally, I had the Physical Therapy Department send over this sample night splint which we often use to correct foot problems. For instance, if a child's left foot turns in, these shoes are connected so that the left foot turns out, or if you have a child with marked external rotation in both lower extremities, tightness of external muscles of the hip, then have the shoes connected with the feet turning in. Sometimes we see a

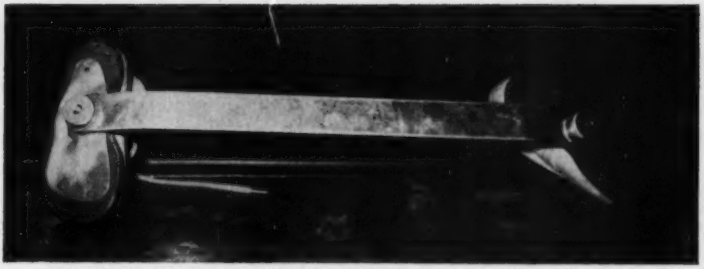

FIG. 1. "Night Splints" as applied to shoes for patient with unilateral adductor deformity

child with a windblown appearance, the right foot turns out, the left foot turns in. With the shoes, we just reverse it. We also use these shoes on polio patients just to keep their feet in a neutral position.

Dr. Anderson:

What about loafers?

Dr. Tobin:

Because of popular demand, especially by the "teenage group", it is hard to get away from them. The youngsters certainly would be just as well off if they went barefooted. The only thing the loafers do is to keep the feet clean as the patient walks. The same thing applies to "Keds", but considering the whole child, and assuming he has a normal foot, you have to make compromises. One point which must be brought up, and I think the pediatricians appreciate it more than the orthopedic surgeons is that in treating these children who have definite deformities and who do require plaster casts, the difficulty at times is treating the mother and not the child. You have to convince the mother that such treatment is necessary because some of them go into a psychological barrier when you say that little Johnnie has to have a cast on his foot. You pediatricians meet that every day.

Dr. Burke:

During the war the Walter Reed orthopedic group were very interested and active in the study of pronated feet in children. I believe Dr. Kite himself was stationed at Walter Reed during this period of time and his influence was reflected in our orthopedic clinics at Children's Hospital. The feet of many children were x-rayed by our department under a standard technique which Dr. Kite had advocated: parallel lines were projected through the astragalus and calcaneus to measure deviations in the relation-

ship of these to the metatarsal bones as an objective method of diagnosis and assaying the correction of adductor varus after cast application. This technique has not been requested of the x-ray department in the past few years. It seemed a very creditable method of following these children at that time but apparently has fallen into discard.

Dr. Tobin:

I think this is of more academic than practical significance and one can usually tell clinically whether these are going in the right direction without using the unlimited x-ray facilities at Walter Reed. It is an interesting study.

Dr. Burke:

That is true. Although the point use to be stressed that the lateral view of the foot during weight bearing was a very important diagnostic factor, yet these babies were under a year old and very few had even supported any weight. Also, as opposed to the casting which Dr. Kite introduced, the Dennis Brown splint has become more popular. I would like to hear a comment from an orthopedist on just when the Dennis Brown splint should be used. Considering that this mild degree of adductor varus with bowlegs is the normal physiologic thing, which corrects in normal childhood development, at what point do these gimmicks become necessary?

Dr. Lloyd:

If the individual case is of sufficient degree to necessitate treatment, we treat them. We determine that only with the fingers and the eyes, there is no mathematical rule we can give.

I do want to clarify one thing if I may. Dr. Anderson apparently misunderstood me slightly, because he said he was unable to evaluate these pronated feet until they were two years old, the implication being that I was able to evaluate them earlier than that. I want you to understand that I can not evaluate them when they are 2 years old, 2½ years old, or in some cases even 6½ years old. When they come in with a pronated heel, if they are as young as 1 year old and already walking, the reasons all these children are treated with an innocuous correction are that in the first place they would never have come in if the mother had not had a question in her mind about that child, and secondly, I do that child no harm whatever to put a little heel wedge in that shoe.

Dr. Anderson:

That is what I misunderstood. If you do not think there is something wrong with the foot at a year, why do you treat it?

Dr. Lloyd:

I think that goes into the old question of psychology. If a mother is worrying about one of her children and the doubt has been put in her mind about an orthopedic condition I definitely do not want to give her a brush-off. I want to do something. For many, many people that is very necessary, otherwise they are going on to someone else, and to someone else, and to someone else, and throw away money with the same result. I tell the parents I am unable to determine at that time whether this foot is going to be a good foot, but I can not possibly do any harm by putting this wedge in here which puts the foot in a more physiologic position. We will follow this child and later on if it becomes necessary to use further treatment or no treatment, we do it.

Dr. Burke:

There are two main objections to that type of philosophy, I would think. It makes the child a patient instead of a normal child, and also the wedge raises the price of the shoe.

Dr. Anderson:

I would like to comment on one thing Dr. Tobin said. If the pediatrician thinks that the orthopedist in all probability will put a light cast on, I think it is up to the pediatrician, who knows the family to sit down and explain to them that this is not a club foot and that the orthopedist may put on a small shell cast which would do in several weeks what long months of massage would do, and if you will do that and pave the way, then you do not have this emotional upset that the mother has when the orthopedist first mentions the word "cast".

Dr. Tobin:

Incidentally, there is a place for exercise for feet. There is a place for exercise for every foot that comes out of a cast, and for every case that is treated by the shoes connected together. It is important. With young age groups, the exercise must be passive, that is, done by the mother. It is certainly part of standard treatment in all cases of club feet, forefoot adduction, and calcaneus valgus, which we did not go into today. When the child gets older, then the child himself might do exercises for the feet. I firmly believe, despite any difference of opinion that might exist here, that active exercise in the older children is another adjunct in the treatment of these foot and knee deformities.

Dr. Burke:

In closing, I would like to thank the panel for an informative session. I think we have learned that knowledge of normal development, considera-

tion of the emotional aspects, and mature judgment are essentials in handling these problems.

ERYTHROBLASTOSIS

Weekly Clinical Conference

Sanford L. Leikin, M.D.,* Juan Jimenez, M.D.†

Dr. Leikin:

Before discussing the specific clinical aspects of the case to be presented this morning, I should like to discuss in more general terms the nature of the problems we face in dealing with erythroblastosis.

The first and most immediate problem is that of the anemia. If it is present at birth the newborn may be hydropic because of cardiac failure due to chronic anemia. Death during the first day of life is practically always due to this severe anemia and its associated conditions. Treatment may at times require vigorous and heroic efforts to lower venous pressure and to raise red cell mass.

The second problem facing the clinician is the danger of the central nervous system involvement. Clinically this condition is most frequently seen from the second to sixth day of life. These infants have stiffness, opisthotonos, vomiting, irregular respiration, a shrill cephalic cry, lethargy, and they feed very poorly. They may not survive the neonatal period; or after what appears to be an amelioration, a delay in motor development becomes apparent usually by the fourth to sixth month of life. By one year of age they are classified as either spastics or cerebral palsied with the usual discouraging prognosis.

CASE REPORT

Dr. Jimenez:

This infant was born on May 8, 1956 by elective Caesarean section at the thirty-seventh week of gestation because of a family history of erythroblastosis.

Past and family history indicated that the baby's mother was Rh negative, and the father Rh positive and homozygous. This baby was the mother's fifth, the first two having been normal, the third anemic at birth, and the fourth who was a premature, having died within one hour of birth. During this pregnancy the mother's Rh titer rose to 1:256.

Immediately after delivery the baby was active and of normal color. The liver was not palpable, but the spleen edge was 2 cm. below the costal margin. The infant's blood was type O, Rh positive; cord bilirubin was 10 mg. per 100 ml.; hemoglobin

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11 gm. per 100 ml.; hematocrit 33 per cent; nucleated erythrocytes 6 per 100 white cells; and white count with differential normal.

An exchange transfusion 450 cc. of O Rh negative blood was performed through the umbilical vein shortly after birth. The procedure was well tolerated. Serum bilirubin was 17 mg. per 100 ml. before the transfusion and 6 mg. per 100 ml. afterwards.

Twelve hours later at 1:00 A.M. on May 9, the patient was very lethargic and jaundice had increased. Serum bilirubin was 21.4 mg., and hemoglobin 11 gm. Another exchange of 500 cc. was done following which the serum bilirubin was 8.56 mg. At 2:00 P.M. that day the serum bilirubin rose to 21 mg. and later that afternoon to 37.7 mg. A third exchange transfusion was performed with a drop in serum bilirubin to 12.8 mg. per 100 ml. Serum bilirubin at 9:15 P.M. was 18.5 mg. The next morning at 3:00 A.M. the bilirubin had risen to 23.18 mg., and by 10:00 A.M. was 26.52 mg. A fourth exchange transfusion of 500 cc. of blood was accomplished lowering the bilirubin level to 11.96 mg.

At this point the baby was given cortisone 12.5 mg. every 12 hours. At 3:00 P.M., May 10, hemoglobin was 10.6 gm., and serum bilirubin 21.54 mg. At 9:00 P.M. serum bilirubin was 23.72 mg., and at 1:30 A.M., May 11, 27.40 mg. per 100 ml. A fifth exchange transfusion was done following which the serum bilirubin was 14.50 mg. At 12:00 Noon it was 14.2 mg. and at 9:00 P.M., 18.12 mg.

On May 12, the serum bilirubin was up to 20.5 mg., but the baby looked more alert and started to take formula well. Cortisone was discontinued. Jaundice started to fade clinically, and on May 15, the serum bilirubin was 9.4 mg., and hemoglobin 8.4 gm. On May 17, the hematocrit was 20 per cent, and the hemoglobin 8.4 gm. A transfusion of 50 cc of packed cell was given and the baby was discharged.

DISCUSSION

Dr. Leikin:

In the case at hand, the first problem facing us was that of obtaining a live baby with which to work. I might state at this point that an initial titer of the mother's serum was done early in the pregnancy, but was not mentioned in the protocol, and was found to be 1:4 in albumin. In the thirty-sixth week of gestation the mother's serum was examined again and the titer had sharply risen to 1:32 in albumin and 1:256 by indirect Coombs testing. There was also a prozone effect, that is, there was no agglutination in the lower dilutions. This finding carries a more severe prognosis. We also found in the mother's history that she had lost one child one hour after birth with what she described as a bloated stomach which we presume to have been hydrops. We were therefore faced with a bad history in a mother whose titer had risen markedly. Now, we do not advocate the routine early interruption of pregnancy because of erythroblastosis, per se. We all know that prematurity makes the prognosis worse because we see most of the kernicterus in such babies. Even the mother who has given birth to a still born baby will, in three cases out of ten, subsequently give birth to a live baby, even though such a baby will probably be affected severely enough to need treatment. However, in this

instance, because of the history and the sharp rise in titer, we felt that if the pregnancy were to continue very much longer we would end up with a stillbirth. Accordingly, we feel that credit in this situation should go first to the obstetrician who took a vigorous, and we feel, heroic attitude about performing a Caesarean section at that point. He did attempt a medical induction prior to the section without success. Subsequent events justified our impression that, had the pregnancy not been interrupted, we would have ended up with a dead baby. As pointed out in the protocol, immediately after birth, the hemoglobin level of the cord blood was 10.0 gm. per 100 ml., the Coombs test was positive, the white cell count was not remarkable, and the number of erythroblasts was within normal limits. The cord blood also showed a bilirubin content of 10 mg. per 100 ml. While preparations were being made for the exchange transfusion, the baby's bilirubin had risen to 17 mg., a markedly rapid rise indeed. I have not previously seen a baby's bilirubin rise so much in so short a time. Following each exchange the bilirubin level dropped for several hours only to rise subsequently.

In studies we have done at the D. C. General Hospital we have found that within a period of one to four hours following the exchange transfusion, the serum bilirubin usually returns to its original level. We feel that what we are accomplishing with an exchange transfusion, in addition to replacing neutral cells, correcting anemia, and washing out antibodies, is that we are also washing out bilirubin. The bilirubin which is located in the tissues enters the blood stream as soon as the blood level declines and thus raises the level in the blood. The repeated exchange transfusions remove this excess of bilirubin. Let me add parenthetically that each time the bilirubin rose, the baby became lethargic, flaccid, and his cry weaker, but as the transfusion progressed, the cry became more vigorous and by the time the exchange transfusion was over, the baby was again active.

We try to use 20 mg. per 100 ml. as the maximum level of bilirubin in the baby's blood. The reason behind this decision is the close relationship between bilirubin and kernicterus. Chemical analysis of the yellow stain in the nuclei of the brain in kernicterus reveals it to be similar to, if not identical with, bilirubin. It has also been demonstrated experimentally that if mouse brain tissue is placed in a Warburg apparatus and bilirubin is added, oxygen uptake is depressed. Further, early workers in the field found that, clinically, the babies acquiring kernicterus were the very ones who were most markedly jaundiced. Diamond's group found that kernicterus occurred in 18 per cent of infants with bilirubin levels between 16 and 30 mg. per 100 ml. and in 50 per cent of those in whom the levels rose above 30 mg. Further, Drs. Richardson, Diamond, and Allen in Boston have now had 4½ to 5 years experience in handling at least 500 cases of erythroblastosis, and they report that they have not seen a single case of

kernicterus since they have used 20 mg. as the critical level of bilirubin for exchange and re-exchange transfusions. Boggs and Abelson in their series have a 99 per cent recovery rate in patients seen within the first 12 hours using the same criteria. In our own work at D. C. General Hospital on ABO incompatibility using 20 mg. per 100 ml. as our critical level we have not seen a single case of kernicterus develop. When we consider Zuelzer's series in which he found an incidence of 13 per cent of kernicterus in an untreated group we feel that we are on firm ground. If a patient showed evidence of central nervous system effects with less than 20 mg. we would intervene earlier. How long is one to continue re-exchanging? We ourselves wondered with this baby after the fourth transfusion but we persevered knowing that if we could keep the infant from getting brain damage, eventually his liver would be able to excrete this excessive load of bilirubin.

I do not mean to sound too dogmatic and leave the impression that by allowing the bilirubin to stay above 20 mg. per 100 ml. the result is always kernicterus, nor conversely that by keeping the level below 20 mg. can kernicterus always be avoided.

However, the danger of exchange transfusion in skilled hands is much less than the risks of developing kernicterus. A "watch and wait" attitude can bear no fruit in this condition. Cord hemoglobin levels can also be used as a guide, but subsequent venous or capillary levels are not of too much value in deciding on exchange transfusion. Boggs and Abelson feel that determination of heme pigments too, may serve as a guide. We have no personal experience with this and do not feel qualified to pass judgment although their figures appear to be valid.

There may be other factors involved, the exact nature of which remains obscure. Dr. Bessman, who is particularly interested in ammonia levels in the blood, recently discussed two babies who had high blood ammonia levels which rose even higher during the exchange transfusion. One of these babies died subsequently with kernicterus. The other survived but has a type of cerebral damage. He postulates that ammonia is the factor required for permitting bilirubin to enter the cell because of the effect of ammonia on cell permeability. We have now begun our own study on the ammonia level and its possible significance in erythroblastosis, but we have not yet gathered enough data to present any conclusions.

Question:

Do you tend to transfuse prematures at a lower level at all?

Dr. Leikin:

Yes, I would say that generally we do. We do not have a specific figure, we still would use 20 mg., but certainly if there is any doubt in our minds, we go ahead at a lower level, depending on the situation at hand.

Question:

Does the placenta protect these babies from the effects of bilirubinemia?

Dr. Leikin:

Yes, it is felt that the reason the bilirubin is lower at birth is that the placenta is acting as an excretory organ allowing the bilirubin to pass into the mother's circulation.

Question:

How do you feel about maternal titers?

Dr. Leikin:

Anything above 1:64 in albumin is considered a good indication for exchange transfusion, at least in our hands. A rising titer has a lot of significance, also, but it is only a general thing, because many times exceptions have been known. Maturity probably has something to do with the result too. Certainly the obstetrician should anticipate trouble when a titer of 1:32 suddenly goes up to 1:256.

Question:

Would you comment on the use of cortisone?

Dr. Leikin:

Administering this drug to the mother prenatally has been of no proven value. There have been several studies on infants which suggest that it is beneficial. I have followed infants with the drug and without it and do not see any great difference.

Question:

Whom do you cross match?

Dr. Leikin:

If we have the time, we crossmatch the mother as well as the baby, because whatever factors are damaging the baby are in higher titer in the blood of the mother. Furthermore, we never know what other factor may be involved in addition to the Rh factor.

Question:

You do not expect the baby to match, do you?

Dr. Leikin:

Yes, a routine saline crossmatch is usually compatible.

THE MENINGOCOCCUS*

Sara E. Branham, M.D.†

Our knowledge of meningococcus meningitis is 150 years old. It was in 1805 that Vieusseaux first described an outbreak in Geneva, Switzerland; the next year Danielson and Mann described an epidemic in Medford, Massachusetts; from that time on people were aware of it as a definite disease entity. In 1886 Hirsch wrote a book about it, in which he pointed out the periodicity of its epidemics. A few years later Hedrich gave further information about that periodicity: epidemics usually come in cycles of 8 to 12 years, with an average cycle of about 10 years. We are past due an epidemic at the present time.

The prevalence of meningococcus meningitis in the Registration area from 1900 to 1945 varied rather widely. During World War I there was a sharp epidemic peak. The incidence then fell to an inter-epidemic level until 1928 when a tremendous wave of meningococcus meningitis swept across the country. After that the incidence fell again and remained at a sporadic level until 1935 when a slow rise led to another peak in 1936. Then the incidence went down again until World War II brought another peak in 1945. There has been no real epidemic since then. Not only does epidemic meningococcus meningitis come in periodic waves, but there is also, strikingly, a definite sporadic level upon which the epidemics are superimposed. It seems almost as though we are dealing with two diseases which are epidemiologically different but clinically alike.

Classifications of the meningococcus into serologic groups were worked out during World War I but no epidemiological significance of these groups had been recognized. During the years since 1928 hundreds of meningococcus strains have been "typed", most from epidemic years and fewer from interepidemic times. Before very long it was perfectly obvious that serological Group I was found abundantly only in epidemics and that it was quite rare during interepidemic times.

The impression is that the epidemic cases are easier to treat. That is not because they are milder but because they are alike. An epidemic that is moving fast and spreading swiftly may be due to just one especially invasive strain in the serologic Group I. Although the cases may vary in severity the meningococci involved are apt to be alike and respond to treatment in the same way.

The sporadic cases, such as have been encountered within the last few years, seem to us to be more difficult to treat. This is not because they are

* Discussion derived from weekly conference.

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more or less severe, but because they are different. The cases are usually unrelated to each other and each causative meningococcus strain has its individual differences. Somehow the other groups of meningococci lack the tendency to spread through a population as does Group I.

An example of individual differences in strains of meningococcus may be seen in their response to sulfonamides and to certain antibiotics. As early as 1938 a definite difference was found in the susceptibility of meningococci to sulfanilamide and sulfapyridine when studied in mice. However, very few strains were used. During the outbreak of World War II Phair and Schoenbach made an extensive *in vitro* study in one of the army camps and found no significant differences in susceptibility to sulfonamides among these epidemic strains. Later Dr. Finland at the Boston City Hospital found a tremendous difference among cultures from sporadic cases, none of which were due to Group I. Among strains from cases in Children's Hospital during 1953 and 1954 there was a 16 fold variation in sensitivity to sulfadiazine. Such variation in susceptibility to sulfonamides does not in any way contraindicate the use of these drugs in treatment. However, it does show that some of these strains from sporadic cases are more resistant than others. It is an innate and not an acquired resistance, and is an example of individual variations among meningococcus strains.

A recent study of the meningococcus strains collected at Childern's Hospital included cultures from 33 cases. Among these there were no Group I strains at all, but only Groups II and II *alpha*, about equally distributed. Apparently there was no constant difference in severity of illness produced by the latter two groups. The number of deaths from Groups II and II *alpha* was equal; there was also an equal distribution of fulminating cases.

A tendency to dread Group I meningococcus infections more than those due to other Groups is no doubt due to the dramatic epidemics that sweep through the country. Although not usually involved in epidemics, Groups II and II *alpha* can cause infections just as severe as those caused by Group I. They are, moreover, often more variable in their response to various agents used in treatment. That in itself is a very important thing.

The Waterhouse-Friderichsen syndrome has usually been attributed to Group I, but, if nothing else has been learned from this study, it is to recognize the potentialities of Groups II and II *alpha* in this role. Actually, then, the serological type of the infecting meningococcus, although of great importance in some ways, is not the chief concern in treating the individual patient, nor is, necessarily, the sensitivity of the microorganism involved. The important thing is getting those agents that are so useful in treatment into the patient in time to do some good. In the fulminating cases which die within a few hours after onset, the tragic thing is that the strains of men-

ingococcus recovered from them are usually very sensitive to all of the antibacterial agents.

Several attempts have been made in the past to develop immunizing agents; they have been so far completely unsuccessful. Vaccines and toxins prepared from the meningococcus are highly toxic and produce severe reactions. Even if a vaccine were available, who should be immunized? Certainly during interepidemic times cases are not frequent enough to indicate vaccination of large groups.

Determination of susceptibility has been attempted by skin testing with meningococcus toxins. In such a study at the National Training School for Boys it was found that about half of the boys gave a positive skin reaction and half did not. Interpretation of these results was puzzling. Does a negative test indicate immunity, as in a Schick test? Or is it like a tuberculin test where a positive test means previous experience with *Mycobacterium tuberculosis?*

As mentioned above, our next epidemic of meningitis is overdue. It is hoped that the vast epidemics have been wiped out by our new methods of treatment. Usually they are heralded by the appearance of Group I strains. Two such strains have been found recently and they came from the same area. It is very important that meningococci should be typed if at all possible, as it is a most valuable procedure in determining epidemiological trends. Typing may not be a necessity for the treatment of an individual case, but, nevertheless, all the knowledge we have about meningococci has come from serological typing.

DIAGNOSTIC X-RAY BRIEF

Isidore Lattman, M.D.,* Joseph M. LoPresti, M.D.†

INCREASED INTRACRANIAL PRESSURE

The roentgenographic changes produced in the calvarium by chronic increased intracranial pressure appear late. These changes which may be observed on flat films of the skull are:

1) Accentuation of the convolution markings (pressor digitoni).
2) Alterations in the sella turcica.
3) Separation of the cranial sutures.

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Although singly, none of these changes is pathognomonic, a combination of two or more of them indicates a definite diagnosis.

An accentuation of the convolutional markings (pressor digitoni) of the calvarium is seen frequently when chronic increased intracranial pressure is present. This gives the skull a "hammered silver" appearance. By itself this change is not diagnostic of increased intracranial pressure, and many normal children will show prominent convolutional markings. However, when in addition to a "hammered silver" appearance, the skull films show alterations in the sella turcica, chronic increased intracranial pressure may be presumed to be present.

The size and contour of the sella turcica in children present numerous normal variations. However, chronic increased intracranial pressure, particularly when it is associated with a dilated third ventricle, may produce alterations in the sella turcica. The floor of the fossa usually is not involved. Most frequently the posterior clinoid processes and the dorsum sellae become rarified and partially destroyed. Occasionally, in severe cases there may be enlargement of the pituitary fossa.

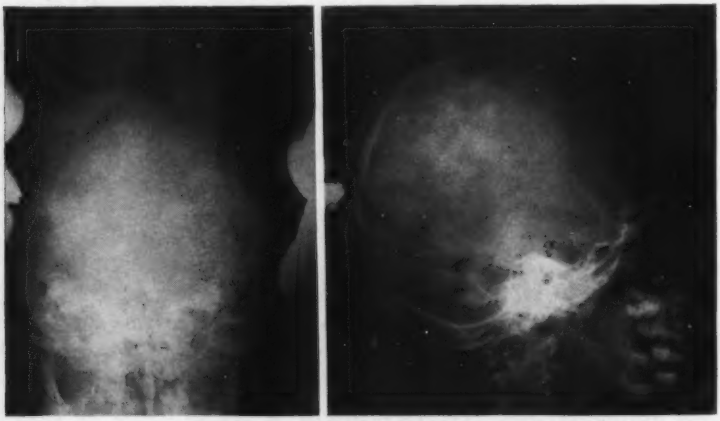

FIG. 1. AP and lateral views of the skull show the characteristic changes of chronic increased intracranial pressure. The convolutional markings are accentuated (pressor digitoni). The coronal and sagittal sutures are separated. The pituitary fossa is enlarged. The clinoid processes and dorsum sellae are rarified and partially destroyed (arrows).

The cranial sutures fuse when children are between the ages of 6 and 9 months. However, ossification of the sutures does not occur until the adolescent period. Therefore, any marked increase in the intracranial pressure can separate the sutures and provide a valuable x-ray finding.

This may be the only change present. All of the sutures may be separated, but in our experience the coronal and lambdoidal sutures are the ones most frequently affected. When separation of the cranial sutures is present to a marked degree, the head will be enlarged.

Finally, it is important for the physician to recognize that chronic increased intracranial pressure may exist in a patient with no demonstrable change in the flat films of the skull. Therefore, the early recognition of this symptom must depend on the history and physical findings. If x-ray changes are found they indicate a process which has been present for some time.

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